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Aspiration Pneumothorax.—Pneumothorax by aspiration, which is induced by allowing filtered air to be sucked in by the negative intrapleural pressure, has recently been advocated by Sorgo, but was devised in 1917 by Forlanini's pupil, F. Morelli. Morelli's technique and a simple apparatus are described. Collapse of the lung by aspiration of air is the most logical method because it is based on physiological principles of respiration. It has been used by Morelli and many other Italian clinicians with excellent results in many thousands of cases. The incidence of effusions with this method is remarkably low.—*Ueber Aspirationpneumothorax, G. Luzzatto-Fegiz, Wien. klin. Wchnschr., September 16, 1932, vi, 1141.*—(*K. F.*)

Treatment of Hilum Cavities by Artificial Pneumothorax.—Peripherally located cavities are well influenced by artificial pneumothorax, because the tension on the cavity wall is released. Cavities which are located near the hilum are not so influenced because the larger bronchi prevent a collapse of the cavity, and because the tension from the diaphragm remains unchanged. For this reason unsatisfactory results have frequently been reported on hilar cavities. It is advised that pneumothorax treatment should be preceded by phrenicectomy or phrenicectomy alone should be done.—*Die chirurgische Behandlung der hilusnahen Kaverne, T. Plieninger, Beitr. z. Klin. d. Tuberk., August, 1932, lxxx, 291.*—(*M. P.*)

Metabolism in Artificial Pneumothorax.—In a fair number of patients with recent and old artificial pneumothorax, a complete study of the gaseous metabolism, with and without exercise, has been done. It was found that the vital capacity decreases with the inflation of air, but that its decrease is less than the air injected. The equivalent of ventilation (a numerical expression of the relation between the respiratory volume per minute and the oxygen consumption) is increased after pneumothorax and returns slowly to normal. The basal metabolic rate is usually not altered by a recent pneumothorax, but after a longer duration the basal metabolic rate is reduced to normal if it was elevated before the treatment. It is concluded that the increase in basal metabolism in pulmonary tuberculosis is not caused by a deficient oxygen supply, but is due to the infectious disease as such. There is no direct relation between vital capacity and basal metabolic rate. Under exercise patients with pneumothorax show a decrease of oxygen consumption and a better utilization. In other words, the gaseous metabolism is improved by pneumothorax. This is proved, too, by the fact that the patient with pneumothorax returns more quickly to a normal gaseous exchange than the patient without pneumothorax, and that the carbon-dioxide excretion is improved. The cause of the carbon-dioxide retention in pulmonary tuberculosis is not a decrease of the respiratory surface, but a greater accumulation of

carbon dioxide in the tissues. Another important cause for the carbon dioxide retention is the fact that blood circulates through diseased tissue in which it cannot make the normal oxygen-carbon-dioxide exchange. When these parts of the lung are collapsed, this inefficient part of the circulation is decreased or obliterated, which explains the observation that the carbon-dioxide excretion is improved after pneumothorax; also the respiratory surface is decreased.—*Ventilation, Grundumsatz und Arbeitsstoffwechsel bei Lungentuberkulose mit künstlichem Pneumothorax, J. Herms and I. Rüttgers, Beitr. z. Klin. d. Tuberk., August, 1932, lxxx, 405.*—(*M. P.*)

Mediastinum in Artificial Pneumothorax.—The condition of the mediastinum is of some importance in pneumothorax work. The mediastinum may shift as a whole, and in such cases the compression of the lung may become insufficient, or the bulging to the contralateral side may embarrass the respiratory mechanism. A slight shift to the contralateral side may have a beneficial effect on contralateral lesions. In many cases a localized mediastinal shift occurs in two typical locations, namely in the upper anterior and in the lower posterior mediastinum. These conditions are called mediastinal hernia, *distension de cul-de-sac pleural* or pneumatocele. In the author's own experience 31 cases of mediastinal hernia were observed, thirty of which occurred in the upper anterior space and one in the lower posterior. The hernias occurred sixteen times in left, eleven times in right and three times in bilateral pneumothorax. The majority of the patients were between twenty and thirty years old. The diagnosis is made most easily by fluoroscopic examination. On roentgen films, the condition may be obscured, because the films are usually taken in maximal inspiration, and in this position the hernia may be less evident than in expiration. Gilbert has reported that the introduction of gomenol oil into the pleural cavity will stiffen the mediastinal structures. A marked mediastinal shift may decrease the vital capacity out of proportion with the inflated air.—*Das Mediastinum bei künstlichen Pneumothorax, E. Hager and F. Langebeckmann, Beitr. z. Klin. d. Tuberk., August, 1932, lxxx, 419.*—(*M. P.*)

Axillary Temperature in Artificial Pneumothorax.—It is well known that the axillary temperature in monolateral tuberculosis is highest on the affected side and it has been observed that with the institution of a successful artificial pneumothorax, the axillary temperature on both sides becomes equal. During the course of collapse therapy an elevation of temperature in the axilla of the contralateral side indicates the onset of a complication even before recognizable clinical evidences appear. The recording of biaxillary temperature is recommended in tuberculous patients receiving artificial-pneumothorax therapy.—*La temperatura biascellare nei fatti controlaterali al pneumotorace terapeutico, D. Canzio, Tubercolosi, January, 1932, xxiv, 1.*—(*U. E. Z.*)

Cardiac Output in Artificial Pneumothorax.—Studies of the cardiac output and other functions of the circulation have been made before and after artificial pneumothorax in 3 cases of pulmonary tuberculosis. The finding of particular interest in this study was the consistent and progressive lowering of cardiac output that took place in all 3 cases as the establishment of unilateral pneumothorax progressed. There was a corresponding increase in the arteriovenous carbon-dioxide (and oxygen) differences. In the two cases in which blood studies were made, there was a fairly satisfactory correspondence between alveolar and arterial carbon-dioxide values. In one case the venous pressure did not change appreciably during the course of the pneumothorax. The arterial blood-pressure showed no consistent variation in any of the 3 cases. There were no significant alterations in the total oxygen consumption or the dioxide output. The pulmonary ventilation increased only slightly toward the end of the series of insufflations. The vital capacity and residual-air volumes diminished progressively.—*Cardiac Output Following Artificial Pneumothorax in Man, D. R. Richards, C. B. Bailey and M. Hiscock, Arch. Int. Med., June, 1932, xxix, 994.*—(*W. I. W.*)

Specific Gravity under Artificial Pneumothorax.—The specific gravity of patients with artificial pneumothorax was determined according to the method of Jamin and Müller. In every case the specific gravity decreased after pneumothorax. The decrease in the specific gravity is caused by the inspiration position of the thorax, and it explains the well-known fact that in pneumothorax the decrease in vital capacity is less than the amount of air inflated.—*Das spezifische Gewicht des Menschen unter dem Einflusse der Pneumothoraxtherapie, A. V. v. Frisch and A. Schneiderbaur, Beitr. z. Klin. d. Tuberk., September, 1932, lxxx, 577.*—(*M. P.*)

Physical Signs in Artificial Pneumothorax.—The diagnosis of pneumothorax is nowadays made primarily from the radio-

graphic appearance of the chest and all other methods of physical exploration are unduly neglected. The classical symptoms described by the older clinicians, especially the metallic phenomena, are very seldom found because they are present only if the intrapleural pressures are much higher than those used with the present technique of artificial pneumothorax. Dyspnoea after a refill points to the presence of a spontaneous pneumothorax which has developed after the inflation. Bulging of the mediastinum and displacement of the heart do not necessarily indicate high intrapleural pressures and are of little diagnostic value. Only positive manometric readings and the appearance of metallic phenomena permit us to differentiate the dreaded valvular pneumothorax from the slowly developing traumatic pneumothorax, which latter, on account of its good prognosis, is of no clinical significance and rarely causes any symptoms. The physical signs of artificial pneumothorax with negative final pressures are very indefinite. There may be a distention of the treated side, noticeable only if there is no emphysema on the contralateral side. Lagging of one side on deep inspiration is very suggestive of pneumothorax. The signs elicited by comparative percussion are surprisingly insignificant. One of the most dependable and constant signs is the apparent descent of the lower borders of the lung on the treated side, which is due to the accumulation of air above the diaphragm, a region toward which a partial pneumothorax early, and quite often expands. It is a moot question whether the respiratory displacement of the lower borders of the lung on the pneumothorax side can be ascertained by percussion. More important is the passive displacement on change of position of the patient. In the knee-elbow position the lower borders of the lung are found several centimetres lower than in the sitting or standing position, unless basal adhesions prevent the descent of the lung. On the untreated side a further displacement on deep inspiration which is absent on the pneumothorax side can be revealed by light percussion. The percutory changes caused by artificial pneumothorax in the apical region are characteristic, especially if Jagic's triangle is used for the topographic percussion of the apices. The medial borders of the lung seem to move upward with increasing collapse of the apex, and Kronig's field of resonance appears to be wider than on the contralateral side. The changes revealed by percussion of the heart vary with the side on which pneumothorax has been induced. In left-sided cases the heart is pushed away by artificial pneumothorax from the wall of the chest and the absolulte dulness of the heart disappears very soon unless pleuropericardial adhesions are present. The right-sided pneumothorax displaces the absolute cardiac dulness markedly to the left and a tympanitic note of percussion may be found one to two fingers to the left of the left sternal border. The determination of the position and the size of the heart by percussion is under these circumstances difficult and far from exact. The auscultation of the heart reveals softness or even disappearance of all heart sounds, particularly in left-sided cases, and functional murmurs over the pulmonary artery and aorta, or pericardial friction sounds, the significance of which is a matter of conjecture. The auscultation of the collapsed lung reveals, as a rule, diminished to absent breath-sounds and occasionally râles over the contralateral side after refills. These râles originate in the collapsed lung under a certain tension, and disappear if the intrapleural pressure is reduced by withdrawal of air. The tactile fremitus is of considerable diagnostic value. It is absent only if the lung is completely compressed. In partial pneumothorax the fremitus is sometimes slightly increased over portions of the lung that are still in contact with the wall of the chest. Since adhesions transmit vibrations from the collapsed lung to the surface of the chest the presence of band- or string-like adhesions can be detected by the use of tactile fremitus.—*Zur physikalischen Diagnostik des künstlichen Pneumothorax, A. V. von Frisch, Klin. Wchnschr., September 17, 1932, xxxviii, 1593.*—(*K. F.*)

Oleothorax.—Some experiences with oleothorax in several types of disease are described. The oil employed was U. S. P. paraffin oil which was sterilized by heating. To 95 cc. of this was added 5 cc. of gomenol without further sterilization. The indications for use of oleothorax are given as follows: (1) as an aid to an ineffective pneumothorax; (2) in cases of purulent and nonpurulent tuberculous pleurisy; (3) stabilization of the mediastinum preparatory to or as an adjustment to incomplete thoracoplasty; (4) substitute for pneumothorax for economic reasons; (5) treatment of bronchiectasis for purposes of compression; (6) some cases of spontaneous pneumothorax; (7) substitute for pneumothorax; (8) some cases of small bronchopleural fistula; (9) presence of recurrent serous effusions. Oleothorax is contraindicated in the presence of a large bronchial fistula and during the acute stage of serofibrinous pleurisy. A simple technique for injecting the oil is described. Oleothorax was employed in several patients

with secondarily infected tuberculous pleurisy without beneficial effects. Details of the beneficial effects obtained in several types of disease by the use of oleothorax are presented. In one patient the procedure was employed for the purpose of compression in preparation for thoracoplasty. In another it was for the prevention of secondary infection of a purulent effusion. Use was also made of it for the control of fever in a patient with a pleural effusion secondary to carcinoma of the oesophagus. A fourth group was that of patients with bronchiectasis. Details of these experiences and of others unrecorded convince the author that oleothorax should be employed for purposes of compression and disinfection only in selected cases when other recognized methods fail.—*Some Experiences with Oleothorax, H. C. Ballon, Am. Jour. Surg., April, 1932, xvi, 1.*—(*J. F. B.*)

Thoracoplasty.—In many patients who have undergone thoracoplasty, success is incomplete because the possibilities of collapse have not been exhausted. The success is not simply dependent on the lengths of ribs removed; it is more important to remove pieces of proper length according to the individual findings in the case. The resection must always be closely paravertebral, and it must always provide sufficient space for the scapula to drop into the costal cage. The complete resection of the first rib and the almost total resection of the second, according to Graf, add to the collapse; but this method should be used only if definitely indicated since it complicates the operation and possibly the after-treatment. Anterior rib-resection produces much less collapse than the paravertebral procedure, but it can be of material help as an auxiliary procedure. If one operates in multiple stages, the intervals between operations must be brief, not only because of new bone-formation, but because pleural scar-tissue prevents proper collapse. Proper bandaging of the thorax for eight or ten weeks following the operation is of utmost importance. Several cases, with roentgenograms, are presented in support of the above statements.—*Beitrag zur Thorakoplastik und ihrer Nachbehandlung, W. Curschmann, Beitr. z. Klin. d. Tuberk., May, 1932, lxxx, 37.*—(*M. P.*)

Apical Thoracoplasty.—The total thoracoplasty of Brauer-Sauerbruch has the disadvantage that healthy lung tissue in the lower parts is often collapsed and functionally disabled. Frangenheim and Graf have inaugurated the apical operation, in which the first rib and possibly the second are entirely removed and a partial resection of the third to seventh rib is done. This operation has been done on 25 cases after preliminary phrenic crushing. Two severely ill patients died ten days after operation. Eleven cases became bacillus-free shortly after operation.—*Über Spitzenplastiken, H. Ulrici, Deutsche med. Wchnschr., March 11, 1932, lviii, 411.*—(*H. S. W.*)

Thoracoplasty for Tuberculous Empyema.—The occurrence of empyema in the course of advanced pulmonary tuberculosis initiates a grave era with a hopeless outlook ordinarily, unless radical thoracoplasty can be instituted early. The history of one patient suffering such complications together with the case report in considerable detail is given. The different stages of treatment are outlined and the beneficial effects obtained by operative procedure are recorded.—*Thoracoplastic Surgery in Chronic Tuberculosis with Associated Empyema, W. H. Thearle, Am. Jour. Surg., May, 1932, xvi, 247.*—(*J. F. B.*)

Gastric Changes after Phrenicectomy.—The gastrointestinal disturbances which are sometimes observed following phrenicectomy on the left side are usually temporary and not of grave nature. Marked alterations in the shape and position of the stomach occur rather regularly without producing functional impairment. These morphological changes, roentgenologically demonstrable, are often extreme, but should not be considered as contraindications against phrenicectomy.—*Form- und Lageveränderungen des Magens nach Phrenicusexairese, H. Lichtenstein, Beitr. z. Klin. d. Tuberk., August, 1932, lxxx, 509.*—(*M. P.*)

Phrenicectomy in Sudan.—In the Sudan under primitive conditions in 1926, phrenicectomy was utilized as the only collapse procedure which was practical under the circumstances. No attempt was made to select the cases, as some symptomatic aid was hoped for in even the worst cases. In all, 58 cases were operated upon; 33 of these were followed to January 31, 1931. Only 3 "cures" were recorded, but 9 of those who "improved" may become "cures." Among 23 other cases which were followed for a shorter period there was improvement in symptoms. Two cases of bronchiectasis showed no benefit. In 4 cases bilateral paralysis of the diaphragm was induced. In one a year of fairly normal life was terminated by haemoptysis. The second, in a hopeless condition, died as a result of the operation. The third patient tolerated the operation well, and in the fourth, com-

plete right-sided paralysis, associated with 6-months paralysis on the left, following crushing of the nerve, benefited the patient although tubercle bacilli persisted. In two advanced cases death followed phrenicectomy, and was due to acute pulmonary oedema, which was limited to the nonoperated side in one, as shown by autopsy, and was more severe in the nonoperated side in the other. In one case thoracoplasty was possible because of the improvement which followed a phrenicectomy. The greatest use for the operation is probably in those cases which ordinary methods of rest and diet just fail to control, and in which the slight additional rest may incline the balance toward the healing side.—*Phrenicectomy in the Treatment of Pulmonary Disease, L. O'Shaughnessy, Lancet, October 1, 1932, ccxxiii, 767.*—(*R. D.*)

Apicolysis.—Lauwer has recommended an apicolysis which is performed by resection of the first and second ribs from a horizontal incision in the supraclavicular fossa. It is pointed out that this procedure is very suitable for the completion of pneumothorax which fails to collapse an adherent apical cavity.—*Die Lauwerssche Apikolyse als Ergänzung zum unvollständigen Pneumothorax, W. Kremer, Beitr. z. Klin. d. Tuberk., September, 1932, lxxx, 623.*—(*M. P.*)

Ligation of Pulmonary Vein in Tuberculosis.—In 4 cases with widespread destructive processes the pulmonary vein was ligated. The procedure proved favorable in cases with infiltrated foci as well as in the healing of cavities. In three of the cases the procedure was instituted on the right side and in one case on the left. The principle, like collapse therapy, depends upon producing an artificial hyperaemia of the diseased lung with a chronic stasis. The method is believed to be valuable in healing cavernous pulmonary tuberculosis.—*Die therapeutische Wirkung der Lungenenligatur bei kavernösen Lungentuberkulosen, H. Edel, Zeitschr. f. Tuberk., 1931, lx, 187.*—(*H. J. C.*)

New Method of Pleural Drainage.—To eliminate some of the objectionable features of the methods now employed in securing air-tight pleural drainage, a new method and a new instrument have been devised. The new tube introducer resembles somewhat a long-bladed, hollowed-out nasal speculum. The blades of this instrument, which are about 2 inches long and $\frac{3}{8}$ inch wide, taper distally to form sharp points. Four illustrations of the various manoeuvres with the instrument are shown.—*A New Method of Instituting Air Tight Pleural Drainage, A. S. W. Touroff, Am. J. Surg., August, 1932, xvii, 254.*—(*J. F. B.*)

Paracentesis in Reclining Position.—Paracentesis for effusions complicating artificial pneumothorax is best performed with the patient reclining, head down, and the kidney regions elevated by pillows. The gas rises to the inferior cul-de-sac, and the fluid gravitates toward the axilla. The trocar is inserted as high in the axilla as possible. This method eliminates fatiguing the patient and the possibility of syncope, mitigates the possibility of injuring the lung, assures more complete evacuation of the fluid, and reduces the incidence of tract infections.—*La ponction axillaire en position couchée des pleurésies à grand épanchement at cours du pneumothorax artificiel, E. Ardoin, Rev. d. l. Tuberc., April, 1932, xiii, 429.*—(*A. E. M.*)

Sanocrysin.—Sanocrysin can be used in pulmonary tuberculosis to obtain radical cure or temporary relief. The clinical reactions and the results of treatment depend upon the extent of disease and the dosage. The reactions result from liberation of toxins within the lesions. They are severe if the lesion is "closed" and mild if it is "open." Advanced disease contraindicates sanocrysin therapy because of the severe reactions obtained. Many of the poor results reported are due to insufficient dosage. The dosage should be based upon the nature and extent of the lesion and the weight of the patient. The first dose is usually 0.1 gm. per kgm. body weight. The first two or three doses are given at intervals of two days and the others at intervals of four, five, or six days. The treatment is continued until there is no reaction after doses of 0.15 or 0.2 gm. per kgm. and is then followed by sanatorium regimen. Better results are obtained by large doses which cause reactions. The combination of sanocrysin with serotherapy has been abandoned. A larger number of smaller doses serves only to increase the amount of metal stored in the body without securing the proper therapeutic result. Reactions are desirable in that activation of a tuberculous focus may be important for later healing. The total dose should not exceed 6 or 8 gm. in order to avoid storage. Three uncontrolled cases are described to illustrate three effects, as follows: (1) the importance of activation of the lesion in "closed" tuberculosis without expectoration; (2) the value of treatment in a case of longer duration with bacilli in the sputum; and (3) the effect of sanocrysin on tuberculous lymph nodes. The results of treatment of

365 cases previously reported by the author are tabulated. Of these, 239 were cases of pulmonary tuberculosis in adults. All of 60 "mild" cases are well. Of 109 "severe" cases 44 are well and 65 improved. Of 70 "very severe" cases 6 are improved and 64 are unimproved. Of 91 cases of pleurisy 86 are well and 5 are improved. All of 18 cases of lymph-node tuberculosis are well. Of 17 cases of pulmonary tuberculosis in children 13 are well and 4 improved. Control cases are not described. *Conclusions:* Early treatment is important. Concurrent use of pneumothorax and phrenicectomy is advantageous. Sanocrysin should be used routinely in tuberculous pleurisy, as it shortens the period of hospitalization.—*Dosage and Results of Sanocrysin Treatment, K. Secher, Lancet, December 19, 1931, ccxxi.*—(*R. M. F.*)

Sanocrysin.—Thirty-six adults with pulmonary tuberculosis were selected for sanocrysin treatment from 320 sanatorium cases treated since August, 1929. This group had failed to improve and was unsuitable for pneumothorax treatment. The average dose of sanocrysin was 0.05 gm. This was increased gradually, with care to avoid reactions, to a maximum dose of 0.75 gm. The total dose was 5 or 6.5 gm. and the average duration of treatment was about thirteen and a half weeks. Eight patients did not complete their treatment either because of the severity of reactions or lack of improvement. One of the eight patients was definitely harmed by the treatment. The sputum was negative in 3 and positive in 25 of the 28 cases in which treatment was completed. The result of treatment was that 22 patients improved and 6 were unimproved. The sputum became negative in 13, or 52 per cent, of the 25 patients who had a positive sputum. There was an average of 20 per cent bacillary loss for patients discharged after sanatorium treatment alone. There was a decrease of 18 per cent in the sedimentation rate of the patients treated with sanocrysin as against 4 per cent for a group of 16 patients undergoing sanatorium treatment alone. Of the 28 patients treated with sanocrysin, 66 per cent lost weight. Fifteen have been followed for a year or more since completing sanocrysin therapy. Of these, 4 are classified as improved, 6 as stationary, 4 as unsatisfactory, and one is dead. Of the 14 survivors, 6 are fit for work and 8 unfit. Of these 14, thirteen had positive and one negative sputum. The sputum of 7 of these became negative after sanocrysin therapy and became positive again in 3 cases. The remaining 7 cases have remained sputum-positive. Sanocrysin is advocated for any case with persistently positive sputum and for any patient in whom induction of artificial pneumothorax has failed. It is used in cases with either exudative or productive lesions. Reactions to the drug were manifested as albuminuria, diarrhoea, vomiting, headache, ulcers of the mouth, a variety of rashes, and obliterative endarteritis with gangrene of the toes. Secher regards the reactions as caused by toxins liberated from the tuberculous focus. The author believes that reactions are due to metallic poisons and cites the experimental work of Oekonomopoulo as proof. Sanocrysin-treated patients who do not improve show a marked increase in sedimentation rate. More rapid improvement is shown in roentgenograms of patients treated with sanocrysin than in those who have sanatorium treatment alone.—*Sanocrysin in Pulmonary Tuberculosis, E. H. A. Pask, Lancet, December 19, 1931, ccxxi, 1346.*—(*R. M. F.*)

Sanocrysin.—This paper deals with 153 tuberculosis patients who were treated with sanocrysin in Brompton Hospital during the years 1926–1928. While the usual indication for sanocrysin was in patients not improving with conservative treatment and yet unsuitable for collapse therapy, it was also used in conjunction with artificial pneumothorax, phrenic evulsion, and thoracoplasty. In order to investigate the claim that sanocrysin is more effective in the recent, exudative type of disease than in the fibrotic, the cases were classified into 9 subdivisions of each of these two main groups, according to the extent of their disease and the severity of their symptoms. The majority of both groups fell into the "3(a)" classification which is listed here as denoting disease more extensive than "2" (disease of slight severity involving the volume of one lobe, or of greater severity involving the volume of half a lobe) and with considerable excavation, and as signifying that the patient is febrile at rest. While large doses were used in the first cases it was later felt that each case must be considered individually, and there was a definite tendency to use smaller doses. The aim was to produce moderate reaction and avoid strong reactions as far as possible. An interval of at least a week was allowed between injections, the total time taken to complete a course of 4 to 6 gm. depending entirely on the degree of reaction produced and the subsequent spacing and increased rate of doses. The list of reactions shows rise in temperature to be the most frequent (about 80 per cent). The frequency of other reactions was: albuminuria 27.5 per cent,

skin reactions 20 per cent, affections of mucous membranes 19 per cent, gastrointestinal symptoms 69.5 per cent, and focal reactions 4 per cent. Of 108 cases with positive sputum, 49 became negative during treatment. In 5 cases the sputum became positive during treatment. In 9 cases the sputum was negative throughout, while in 36 there was no record. Of the 49 who became negative, 29 were known to have become positive again within three months. In assessing the immediate results four factors were considered: (1) disappearance of bacilli from the sputum; (2) symptomatic improvement; (3) improvement in physical signs; and (4) X-ray changes interpreted as indicating healing. Considering the whole group on this basis 16 per cent were much improved and 51.5 per cent improved. The relative results of the two main groups showed 27.5 per cent much improved of those with exudative type of disease, contrasting with 6 per cent of those with the chronic type, and 47 per cent improved contrasting with 54 per cent. In a series of 18 cases sanocrysin was used in connection with artificial pneumothorax with results slightly better than those quoted for the group as a whole. The results in 5 cases that received thoracoplasty suggest that sanocrysin has a value, either in preparing a patient for this operation, or as treatment for the better side. There is need for controlled investigation, particularly of the effect of large doses on early cases showing a relatively slight degree of lung involvement.—*On the Use of Sanocrysin in Pulmonary Tuberculosis, H. E. Mansell, Lancet, October 15, 1932, ccxxiii, 837.*—(*E. H. K.*)

Skin Changes from Sanocrysin.—*Chrysiasis* denotes changes in the skin of patients treated with sanocrysin, and results from the deposit of gold. Similar to *argyria* the exposed parts of the body (face, neck and hands) have a bluish or steel-gray tint. The development of Chrysiasis is proportionate to the amount of sanocrysin used. When the amount was less than 5 cgm. per kg. body weight no changes occurred, but in cases receiving over 15 cgm. none was negative. Before the development of the skin changes an interval of 1 to 3 years may elapse. A regression of the pigmentation was not observed.—*Über das Auftreten von Chrysiasis bei früher mit Natriumaurothiosulfat behandelten Lungentuberkulösen, J. N. Lorenzen, Beitr. z. Klin. d. Tuberk., 1931, lxxvi, 686.*—(*H. J. C.*)

Metal-Salt Therapy in Guinea Pigs.—Metal-salt solutions of manganese and cadmium, which had previously yielded results in rabbits, proved valuable in guinea pigs, in that about half of the animals showed complete healing.—*Metallsalztherapie. (Behandlung von experimenteller Meerschweinchentuberkulose.), L. E. Walbum, Zeitschr. f. Tuberk., 1931, lx, 204.*—(*H. J. C.*)

Iodine in Tuberculosis.—A review of the recent literature shows that opinions are divided as to the therapeutic indications and contraindications of iodine medication in tuberculosis. It is well established that iodine accumulates more in tuberculous foci than in normal tissues, as it does in other diseased tissues. It seems probable that iodine combines with certain toxic products, and exerts therefore a detoxifying action. This might explain some of the favorable results reported with iodine medication. Fifty-two far-advanced patients were treated with simple iodine salts, and especially with *endojodin*, a complicated organic compound, which contains 0.118 gm. of iodine per cc. The following beneficial effects were observed: subjective improvement of the general condition, greater ease of expectoration and of respiration, increase in weight, decline of temperature, reduction of the sedimentation rate, and increase of haemoglobin, erythrocytes and lymphocytes. Following large doses, the number of bacilli in the sputum decreases noticeably.—*Zur Jodbehandlung der Lungentuberkulose, A. Sylla, Beitr. z. Klin. d. Tuberk., May, 1932, lxxx, 51.*—(*M. P.*)

Iodine in Experimental Tuberculosis.—In the present study an attempt was made to determine principally the possibility of injecting elemental iodine into the animal body and, if practicable, to use it in the treatment for tuberculosis. Three rabbits received intravenously amounts that were equivalent to 10, 20 and 30 mgm. of free iodine per kg. body weight. All three survived the injection. The rabbit receiving 30 mgm. showed more apparent uneasiness during the twenty-four hours following the administration of iodine. The results obtained in therapeutic experiments show that the small dose (2 mgm. per kg. body weight in most of the experiments) was not effective, probably because it was quickly neutralized by the alkali reserve of the blood. A dose of 30 mgm. of iodine per kg. body weight does not kill a normal animal, but one of 20 mgm. per kg. is fatal to a rabbit with severe miliary tuberculosis. It is thought that this subject deserves further consideration under more appropriate conditions.—*The Use of Iodine and of Certain Iodine Com-*

pounds in Experimental Tuberculosis, G. Knaysi, J. Infect. Dis., March, 1932, l, 261.—(F. G. P.)

Diathermy in Pleural Adhesions.—Localized diathermy treatment for pleural adhesions may produce a loosening-up of the adhesions, atrophy and stretching. This may help materially in collapsing such cavities as are held open by adhesions. In particularly favorable cases it may be possible to sever adhesions by this method.—*Beobachtungen über die Wirkung von Diathermieströmen auf strangförmige Pneumothoraxadhäsionen und die durch diese ausgespannt gehaltenen Kavernen, H. Wiercinski, Beitr. z. Klin. d. Tuberk., August, 1932, lxxx, 447.—(M. P.)*

Heliotherapy in Pulmonary Tuberculosis.—The literature is reviewed on the use of light in the treatment of pulmonary tuberculosis, and the diversity of opinion as to its merits is brought out. While certain competent observers are convinced that they have had good results in selected cases, the dangers of the method, such as reactivation of quiescent disease, the production of haemoptysis, the dissemination of infection, and the production of focal reactions, must always be borne in mind. The difficulties of treatment by light are chiefly two: (1) the estimation of the correct dosage, and (2) the selection of the suitable case. The dosage question can be fairly easily decided, but rules for the selection of the individual case will probably always be a personal matter. The method should be used in pulmonary cases only as an adjunct to the established methods of treatment, and hence it should find no place outside the residential institution.—*The Treatment of Pulmonary Tuberculosis by Light: Phases of the Controversy, E. A. Underwood, Brit. J. Tuberc., October, 1932, xxvi, 178.—(E. H. K.)*

Bactericidal Action of Ultraviolet Light.—Experimental evidence has demonstrated that ultraviolet light has a direct bactericidal effect on bacteria. A suspension of tubercle bacilli, containing 2,750,000 organisms per cubic centimetre, was killed within four minutes by the absorption of 1.42×10^9 energy ergs per cubic centimetre derived from the complete ultraviolet band of a mercury-quartz arc (Kromayer). A like amount of energy destroyed dried tubercle bacilli in the same suspension within four minutes. Exposure of tubercle bacilli in suspension to ultraviolet at 5.93×10^6 ergs per second per cubic centimetre for twenty-five minutes did not alter their acid-fast staining properties. Tubercle bacilli, fixed by heat and then irradiated for five to twenty-five minutes, showed changes indicative of disintegration and irregularities were observed in staining by the Ziehl-Neelsen method.—*The Bactericidal Action of Ultraviolet Irradiations of Tubercle Bacilli, E. Mayer and M. Dworski, Tubercle, August, 1932, xiii, 500.—(M. E. P.)*

Treatment with "AO."—The vaccine *AO*, advocated by Arima, was tried on 11 patients. In some cases focal reactions were observed, and the results were not encouraging.—*Bemerkungen zur Anwendung des "AO," Warnecke and Ritschel, Beitr. z. Klin. d. Tuberk., September, 1932, lxxx, 642.—(M. P.)*

Insulin in Treatment of Tuberculosis.—The value of insulin as a therapeutic measure in certain selected cases of pulmonary tuberculosis to restore normal appetite and combat loss of weight and resistance is outlined. The systematic use of insulin in all cases of tuberculosis is not to be encouraged. It must be rigorously limited to inactive and afebrile cases with clinical, X-ray and bacteriological controls. Haemoptysis, marked hypotension, and marked reactions following the injections contraindicate its use. The maximum daily dose of 30 units should not be exceeded and should not be continued for more than two or three weeks. The initial dose of 5 units should be continued daily for the first week. Thereafter, other things being equal, the dose should be increased by 5 units weekly until the maximum is reached.—*The Use of Insulin in the Treatment of Pulmonary Tuberculosis, P. Ellman, Brit. J. Tuberc., October, 1932, xxvi, 187.—(E. H. K.)*

Hepatosplenotherapy in Tuberculosis.—Extracts of the liver and spleen are physiologically synergistic in activating haematopoietic organs. They stimulate the defense mechanisms of the body against tuberculosis. Their action is also reflected in the reticulo-endothelial system, the vegetative nervous system and upon cholesterin exchange. In Italy, a preparation is available for physicians under the name of *Hepasplenina.—Gli estratti epatosplenici nella cura della tubercolosi, N. Sforza, Tubercolosi, February, 1932, xxiv, 37.—(U. E. Z.)*

Injuries of Thorax.—In injuries of the chest, complications may appear following a free interval. This fact is apparently not generally known; five such cases are therefore

reported. In all, the injury was a blow on the thorax, but the injury was not considered serious except in one case where slight haemoptysis occurred. A fractured rib was present in two cases. Spontaneous pyopneumothorax occurred in three cases, after intervals of 1, 5, and 3½ weeks; empyema appeared in one case 2 weeks after the injury; and in the fifth case severe haemothorax was diagnosed after an interval of 6 weeks. It would seem probable that the blow on the chest-wall caused a contusion or laceration of the lung, with subsequent lobular pneumonia and necrosis permitting rupture into the pleural cavity.—*Injuries of the Thorax: Serious Pulmonary Complications Following a Free Interval, J. Head, Arch. Surg., September, 1932, xxv, 601.—(R. D.)*

Massive Collapse of Lung.—The physical and roentgenographic signs of massive collapse of the lung are described. Various theories concerning the causative mechanism are reviewed. There is disagreement with Briscoe's theory that there is ascent of the diaphragm, with resulting deflation of the lung with the subject in the supine position, because no such difference in the position of the diaphragm has been observed. A case history is summarized, as follows: A male, aged 25, received a severe blow over the twelfth dorsal vertebra. He fell and struck the left side of his chest. There were pain and tenderness at these locations. No abnormal physical signs were found on examination of the lungs. Twenty-four hours later severe dyspnoea and cyanosis, pain in the left chest, fever, accelerated pulse-rate, cough and mucoid expectoration developed suddenly. The typical signs of massive collapse of the left lung were present. These were confirmed roentgenographically. A roentgenogram of the dorsal vertebrae revealed no fracture. The atelectasis disappeared completely within eleven days. Eighteen days later the patient was admitted with a fracture-dislocation involving the second and third lumbar vertebrae. Most of the damage was at the left anterolateral part of the second lumbar vertebra, which is the region where the left crus of the diaphragm originates. The sympathetic chain pierces the crus. The massive collapse of the lung is explained on the assumption of injury to the sympathetic, with collapse resulting from disturbance of the balance between the sympathetic and vagus innervation of the lungs, as suggested by Morison.—*Massive Collapse of the Lung with Notes on a Post-Traumatic Case, T. E. Gumpert and D. MacLennan, Lancet, November 7, 1931, ccxxi, 1017.*—(*R. M. F.*)

Absorption of Air in Atelectasis.—Serial experimental studies give evidence that atelectasis is the end-result of interchange between the gases of the alveoli and the perialveolar capillary blood through the pulmonary endothelium. Atelectasis must inevitably follow complete bronchial obstruction as a result of the absorption of the alveolar gases; conversely atelectasis cannot occur unless the alveolar gases are completely shut off from the external air. The most important factor in the accomplishment and regulation of this exchange of gases is the difference in partial pressures of the gases in the alveoli and perialveolar capillary blood. Graphic representation of percentages of oxygen and carbon dioxide obtained by alveolar gas analysis before obstruction and for a period of over 10 hours after obstruction are given. Within 10 minutes after obstruction the oxygen has fallen from 15 to 5 per cent and the carbon dioxide has gone from 5 to 6 per cent, showing that an approximate equilibrium with the venous alveolar capillaries has been established. From this point on, a series of gas exchanges takes place, leading ultimately, in 12 to 16 hours, to atelectasis. Quantitatively, the entrapped alveolar gases pass through the respiratory membrane into the blood circulating in the perialveolar capillaries until airlessness of the involved area is produced. Qualitatively, the percentages and partial pressures of the gases comprising the alveolar air tend to, but never quite, reach an equilibrium with the gases of the venous blood. The mechanism of production of atelectasis in the compressed lung (pneumothorax, pleural exudate, intrathoracic tumors, etc.) is exactly the same as in bronchial obstruction.—*Studies in Pulmonary Gas Absorption in Bronchial Obstruction, III. A Theory of Air Absorption in Atelectasis, P. N. Coryllos and G. L. Birnbaum, Am. J. M. Sc., March, 1932, clxxxiii, 347.*—(*W. I. W.*)

Internal Drainage and Massive Collapse.—The cause and entire clinical picture of postoperative massive collapse of the lung (pulmonary atelectasis) is explained on a combined mechanical and infectious basis. Postoperative pulmonary atelectasis is due primarily to the bronchial obstruction resulting from the presence of excess intrabronchial secretion and the influence of "internal drainage" upon these secretions. By "internal drainage" is meant the spilling of material from place to place within the tracheobronchial tree to spread infection, obstruct bronchi, and shut off the air-flow to a portion of the lung. This spilling depends on the amount and viscosity of the

secretion and the posture of the patient. In certain pulmonary diseases the excess secretions are already present within the tracheobronchial tree before the operation; but, in other conditions, the secretions are aspirated from the mouth and nasopharynx during or immediately following the operation. Internal drainage accounts for the varied onset of postoperative pulmonary atelectasis, diversity of symptoms, predilection of sites, extensiveness, migrations, recurrences, and the response to various treatments. The vasomotor, phrenic, vagus or sympathetic reflexes, angioneurotic oedema or compression of the chest-wall are not considered as primary aetiological factors in postoperative atelectasis. Careful attention to the mouth, hygiene, the posture of the patient during and following the operation, the prevention of aspiration and the removal of intrabronchial secretions should either prevent atelectasis and other postoperative pulmonary complications or decidedly lessen the frequency of their occurrence. Postural exercises, carbon-dioxide inhalations and bronchoscopic treatments have proved of value in the management of patients with postoperative atelectasis when carried out in accordance with the principles of internal drainage.—*Internal Drainage. A Factor in the Production of Postoperative Massive Collapse of the Lung (Pulmonary Atelectasis). Suggestions as to Prevention and Treatment. W. B. Faulkner, Jr., and E. C. Faulkner, Am. J. M. Sc., September, 1932, clxxxiv, 370.*—(*W. I. W.*)

Sclerosis of Pulmonary Artery.—A review of the literature shows that there is general agreement that sclerosis of the pulmonary artery may be secondary to changes in the heart and lungs, such as mitral disease, emphysema, pleural adhesions, and chronic bronchitis, and may also be incidental to generalized arteriosclerosis. When sclerosis results from these conditions it is the main trunk and larger branches which are affected. The changes are as a rule without clinical significance or are overshadowed by symptoms that are produced by the primary lesion. Primary sclerosis, a separate entity, affects the smaller branches of the pulmonary artery and is obliterative in type. This condition is rare and may be overlooked clinically. It occurs before the sixth decade and more commonly in males. The signs and symptoms include cyanosis, polycythaemia, dyspnoea, haemoptysis, somnolence, vertigo, and heart pain; anasarca and passive congestion of the liver, spleen, kidneys and intestines follow; dilatation of the right side of the heart and of the trunk of the pulmonary are evident. Syphilis, rheumatism, and the inhalation of gases have been considered as aetiological factors. A negro male, aged 40, was admitted complaining of shortness of breath, cough, haemoptysis, dizziness, and a swelling of the legs. The cardiac decompensation was relieved and he was discharged. Over a period of 5 years he was readmitted 13 times with a similar clinical picture. Polycythaemia was a fairly constant finding, and cyanosis was recorded 3 times. The Wassermann reaction varied from 4 plus to one plus. He received 3 doses of 0.6 gm. of neoarsphenamine early in his treatment. Microscopical examination of the small arterioles in the lungs showed them to be tortuous and thickened, the latter due to an increase in the media, both muscle and elastic tissue being hypertrophied. The lumina were narrowed. In some of the small arteries the intima showed fibrosis and thickening. The connective tissue about the bronchioles was increased, their lumina were narrowed, and folds in the mucosa exaggerated. After prolonged search no spirochaeta pallida were found in the sections from the lungs. Congenital hypertrophy of the media or faulty development may predispose the small pulmonary arteries to the obliterative changes observed in these cases.—*Primary Sclerosis of the Pulmonary Artery, E. R. Pund and T. B. Phinizy, Ann. Int. Med., May, 1932, v, 1391.*—(*R. D.*)

Traumatic Aneurysm of Pulmonary Artery.—Trauma of the pulmonary artery is usually quickly fatal because of haemorrhage, and rarely results in aneurysm. In the case here reported, a bullet entered the thorax in the midsternal region and resulted in an arteriovenous aneurysm of the right pulmonary artery and vein. There was haemoptysis at no time except immediately after the injury. Cyanosis and dyspnoea appeared immediately and persisted until death. For four months following the injury there was a right haemothorax with fever. A physical examination nine years after the injury revealed a cutaneous and subcutaneous venous network on the forehead, face and trunk; rapid respiration and slow pulse; slight systolic hypotension; and pain on palpation over the fourth and fifth right intercostal spaces in the parasternal region. A roentgenograph revealed a rounded shadow about the size of a hen's egg located in the juxtahilar paramediastinal region, separated from the heart and large blood-vessels. It expanded with systole. Breath-sounds were diminished over this area. The eyegrounds showed evidence of extraocular venous obstruction. The Wassermann test was positive on the spinal

fluid only. Because of the short circuit between the pulmonary artery and vein, only half the blood reached the lung and was oxygenated. Therefore the oxygen saturation of the arterial and venous blood was found to be low; and the carbon-dioxide tension of the alveolar air was low, due to hyperventilation of the blood which was able to pass through the lung. The fact that five minutes' exposure to an atmosphere of pure oxygen did not modify the cyanosis confirmed the diagnosis of arteriovenous fistula. The only therapy possible consisted in dietetic and hygienic measures, and antisyphilitic treatment. Death occurred following a right hemiplegia and symptoms of bilateral pulmonary infarction. An autopsy revealed a circular communication, 15 mm. in diameter, between the right pulmonary artery and vein.—*Aneurisme de la branche droite de l'artère pulmonaire, M. R. Castex, A. V. Di Cio and A. Battros, Arch Méd. Chirurg d. l'Appareil Resp., 1931, vi, 303.*—(*E. K.*)

Deviations of Mediastinum.—This paper recapitulates a seven-year clinical study of the significance of, and mechanisms involved in deviations of the mediastinum, a previous report having been published. The term *fibrothorax* includes all permanent mediastinal deviations due to pulmonary lesions. A fibrothorax was diagnosed by clinical and roentgenological methods in 104 of 2,102 noninstitutionalized patients, an incidence of 5 per cent. No autopsy or laboratory data are reported. Each of these 104 patients had proved pulmonary tuberculosis, but not all had cavitation. Thirty-eight were either in the third decade of life or over 45 years of age. At examination 65 were classified as in good general condition, and 39 as in poor or bad. Fifty-nine had unilateral pulmonary tuberculosis, 45 had bilateral lesions. Seventy-five were afebrile at rest and did not exceed 37.8° (rectal?) on exertion. The typical patient with fibrothorax presents himself for examination complaining of spasmodic shortness of breath, palpitation, cough and expectoration, usually of many years' duration. Physical examination reveals signs of hyperventilation of one lung and hypoventilation of the other, with exaggerated transmission of tactile and vocal fremitus, and of the respiratory murmurs, indicative of compression and fibrosis. Of major importance is the auscultation of the tracheal *bruit* lateral to the manubrium and well transmitted throughout the upper lobe, and sometimes exaggerated cavernous breathing below, indicative of a partially collapsed bronchus. Râles may be present or absent, depending upon the stage of evolution of the pulmonary lesion. The cardiac impulse is deviated toward the involved side. On fluoroscopy, inequalities of expansion and density are noted, and the retroaortic space is sometimes clearer than usual. The roentgenogram, which must be of short exposure (0.1 seconds) at a distance of 1.65 metres to 2 metres with hard rays to obtain proper contrast, furnishes the greatest information. Great care must be taken to ensure an accurate anteroposterior placement. The diagnostic points are deviation of the trachea (less frequently the deviated oesophagus can be visualized); visualization of the lower thoracic intervertebral discs normally concealed by the heart; visualization of the paravertebral space on the involved side. Forty-two cases corresponded completely with this description and were classified as spontaneous, complete fibrothorax. Twenty-one had not reached this final stage and were diagnosed as incomplete. Eleven showed deviation of the trachea and aorta only and were termed *partial, superior.* Nine revealed deviation of the heart only and were termed *partial, inferior.* Of 23 cases of artificial fibrothorax, all sequelae of artificial pneumothorax, 15 were total, 1 incomplete, and 7 partial. Eight of these had had complicating effusions. In each subdivision the number of left deviations exceeded those to the right, the total figures being 71 to the left and 35 to the right (*sic*). Four additional cases could not be fitted into this classification, having had artificial pneumothorax superimposed on a preceding fibrothorax. In discussing the mechanism of these deviations exception is taken to the claim of Guinard and Hinault that the pleura plays a dominant rôle. It is shown that in many of the above cases no pleural symphysis exists, and that in one case of complete pleural symphysis no deviation existed. The true dominant factor is a circumfocal pulmonary fibrosis of massive extent which by retraction both pulls over the adjacent mediastinal structures, producing a partial fibrothorax, and, by diminishing the volume of one lung, disturbs the intrathoracic pressure equilibrium, and total fibrothorax results. In confirmation is cited a case of total fibrothorax associated with a purely apical lesion. Incomplete fibrothorax, or its nonoccurrence, is explained by the inability of the contralateral lung to hyperexpand as in a progressive contralateral lesion. In one case on the appearance of a contralateral lesion the fibrothorax reversed itself. This theory fails to account for fibrothorax in the presence of massive effusions, in which case extension of the pleuritis to the paren-

chyma with fibrosis and retraction must be postulated. A possible factor in the selective occurrence of fibrothorax among apparently identical cases is the fact that of 77 cases of the spontaneous type, 51 had an undoubted tuberculous ancestry. In the evolution of the lesion the heart is deviated in short jumps rather than progressively, each shift giving rise to a more or less evident symptom complex of pain, fever, palpitation, and, in extreme cases, decompensation. Almost invariably, however, these crises are benign. The significance of fibrothorax lies in its being an attempt at a spontaneous cure by massive fibrotic obliteration which is often permanent. Of the 104 cases, most under observation more than three years, 18 died, 12 are approaching a fatal termination, and 74 are alive and well, 31 of whom have returned to a normal life. The prognosis is therefore good, and treatment is one of *laisser faire.* Pneumothorax especially, by equalizing the intrathoracic pressure, impedes the fibrothorax, and, except as an emergency measure, as in haemoptyses or sévere cardiac crises, is contraindicated. In confirmation, four cases so treated are cited, three of whom were better before the inception of pneumothorax, bore it poorly, and resumed favorable progress after its cessation. Phrenicectomy and thoracoplasty diminish the hemithoracic volume and are therefore not contraindicated if the slowness of the natural cure renders the outcome doubtful. Measures to aid the fibrosis—rest, tuberculin, gold-salt therapy, small pleural insufflations of air (irritative)—have been successfully employed in conjunction with the usual pthisiotherapeutic measures. In conclusion, the plea is made that careful investigation of the mediastinal position be included in all cases of pulmonary tuberculosis, for this sign has a prognostic value and a therapeutic indication of prime importance.—*Contribution a l'étude des fibrothorax, C. Vincenti, Rev. d. l. Tuberc., April, 1932, xiii, 345.*—(*A. E. M.*)

Biliobronchial Fistula.—Biliobronchial fistula may be defined as a communication between the biliary tract and the bronchial tree which permits bile to ascend into the bronchi. The fifty-third case not caused by echinococcic or amoebic infection is recorded. A white male laborer, 42 years of age, gave a fairly typical history of cholelithiasis. At operation a gall bladder distended with stones and black bile was removed. The cystic duct was obliterated by scar-tissue. The dilated common duct contained no palpable stones. It was drained by incision and insertion of a Horgan tube. The patient improved steadily. The tube was accidentally removed by the patient fifteen days after operation. No other complications occurred. The wound healed except for a small opening at the upper end from which a small amount of bile drained. Except for this annoying drainage, the patient was well for three and a half years. At that time the discharge stopped and the opening healed. Two weeks later he had pain in the lower right chest. After thirty-six hours he began to cough and raised with difficulty 500 cc. of yellowish seropurulent material. Postural drainage could not be employed because of suffocation. An attempt was made to visualize the fistula by the injection of iodized oil through a bronchoscope. Teleoroentgenograms showed an area of increased density, 11 cm. long and 3 mm. wide, extending from the root of the lung to the obliterated costophrenic angle. The subdiaphragmatic abscess was drained through the tract of the old fistula by means of a rubber catheter. By suction 780 cc. of thick creamy pus was obtained. The following day 300 cc. of sodium iodide was injected through the catheter and iodized oil again injected through a bronchoscope. These solutions were shown to merge and their paths of direct union were outlined. The abscess healed and the fistula closed without complications. This case is reported as the first case in which the fistula was demonstrated by lipiodol injections.—*Biliobronchial Fistula, W. R. Laird and W. V. Wilkerson, Am. J. Surg., February, 1932, xv, 317.*—(*J. F. B.*)

Roentgenography of Diaphragmatic Hernia.—A case of diaphragmatic hernia (colon) following a wound of the thorax is reported. For sixteen years the patient went under the diagnosis of thickened pleura. The diagnosis was corrected with the help of the X-ray and contrast filling of the intestines.—*Zur Frage der traumatischen Zwerchfell-Hernie und zur Bedeutung des Roentgenverfahrens für ihren Nachweis, S. Samuelson, Deutsche med. Wchnschr., February 26, 1932, lviii, 332.*—(*H. S. W.*)

Primary Angiofibroma of Diaphragm.—A white male, aged 50 years, was admitted to the Tuberculosis Service of the Philadelphia General Hospital November 12, 1931. His history and clinical course were typically those of extensive pulmonary tuberculosis. Physical examination showed extensive involvement of both lungs and the sputum was positive for tubercle bacilli. No other findings of significance were noted. *Roentgen diagnosis:* Extensive ulcerative fibrocaseous tuberculosis of left lung and upper lobe of right lung, with cavitation of both

upper lobes; atelectasis of left lower lobe; neoplasm arising either from right diaphragmatic pleura or right dome of liver. Fluoroscopic examination revealed this tumor mass to move synchronously with the diaphragm. The patient grew progressively weaker and died December 5, 1931. Necropsy examination confirmed the clinical diagnosis of pulmonary tuberculosis. The shadow described in the roentgenogram was cast by a tumor lying between the leaflets of the right dome of the diaphragm. It measured 12 by 6 by 5 cm., and was irregularly rounded and extremely firm. The cut-surface showed a tough, grayish-white matrix, mottled by many roughly round haemorrhagic areas most marked near the centre. There was a depression in the posterosuperior surface of the right lobe of the liver, thus forming a nest into which the inferior surface of the tumor fitted snugly. Histological studies of this tumor revealed an angiofibroma.—*Primary Angiofibroma of the Diaphragm, E. B. Holmes and W. Brady, Am. J. M. Sc., May, 1932, clxxxiii, 679.*—(*W. I. W.*)

Intrathoracic Dermoid Cyst.—A woman, 30 years of age, complained of repeated and long periods of cough and expectoration. Roentgenological, physical and sputum findings suggested pulmonary gangrene. In a two-stage operation, the pulmonary focus was incised and good drainage was established, producing large amounts of foul material. Three and a half years later, the wound was still wide open and secreted some pus. A deeper incision was made, and a round smooth tumor, the size of a cherry, was visualized. It was adherent to a larger tumor mass, to the pericardium and the mediastinal structure. Only part of the tumor could be removed. Histologically it was a typical dermoid cyst. At a second operation, more of the tumor was excised. After this, the patient improved markedly, the secretion diminished, but the bronchial fistula remained patent so that a plastic operation is considered for the future. Intrathoracic dermoid cysts usually produce no symptoms unless they exert undue pressure or unless they become infected.—*Zur Diagnose und Behandlung der intrathorakalen Dermoidcysten, P. Starcke, Beitr. z. Klin. d. Tuberk., August, 1932, lxxx, 278.*—(*M. P.*)

Congenital Cystic Lung.—A man, 36 years of age, who had always been well until eight years ago, was admitted on account of pulmonary haemorrhages. Eight years ago he had pleuritic pain and was diagnosed as having pulmonary tuberculosis. With the same diagnosis he was treated repeatedly in various hospitals. On admission, he is in good general condition. The right side of the thorax lags and the musculature of the right chest and arm is less developed than on the left side. There are coarse râles over both lungs, and over the lower right the breath-sounds are much diminished. Tubercle bacilli cannot be found in the scanty sputum. A roentgenogram of the chest shows honeycombing of the upper right, marked deviation of the trachea to the right and some cyst-like shadows in the lower right lung field which is otherwise devoid of markings. A bronchogram shows the honeycombed areas filled, and multiple cysts with fluid levels near the hilum and in the lower right field. In the midlung field, a few bronchi are seen which terminate abruptly without much branching. The largest cavity is the size of a walnut. Since the cysts seem not to be infected according to the clinical picture, active surgical therapy, the only possible therapeutic measure, seems not indicated.—*Ein Fall von angeborener Cystenlunge, M. Bönniger, Beitr. z. Klin. d. Tuberk., May, 1932, lxxx, 132.*—(*M. P.*)

Cystic Disease of Lung.—Three cases of cystic formations in the lung are described in some detail. The lesions were roentgenologically demonstrable. In all cases the surrounding tissue did not show any alteration that would indicate a destructive or inflammatory process. For this reason, tuberculosis, abscess, gangrene and tumor can be excluded. Cystic formations may be emphysematous blebs, blebs due to scar-tissue, or congenital formations. Following trauma, cyst-like formations may be produced by interstitial emphysema, which, then, may cause spontaneous pneumothorax. Congenital cysts form in some cases the basis for bronchiectasis.—*Beiträge zur Klinik cystischer Lungenveränderungen, H. Siems, Beitr. z. Klin. d. Tuberk., September, 1932, lxxx, 655*—(*M. P.*)

Miliary Lung Disease.—A clinic at Picher, Oklahoma, for the study and control of silicosis and tuberculosis among the miners is maintained by the United States Bureau of Mines, the Metropolitan Life Insurance Co., and the Tri-State Zinc and Lead Ore Producers Association. Physical examination, including roentgen examination of the chest, is made of the men prior to employment and at least once yearly thereafter. More than 18,000 individuals have been examined. About 125 cases of typical miliary lung disease are described as having been found by roentgen examina-

tion among this group. The cases ranged in age from 16 to 69 years. The greatest number of cases occurred between 20 and 30 years of age, and the next greatest between 30 and 40 years. These figures indicate that the greatest number of cases occurs in the years in which adult tuberculosis is most common. The past occupations given by the men revealed that 54.4 per cent had been farmers, 12.8 per cent had been in school and 31.2 per cent had been oil-field workers, pulp-mill workers, mechanics and teamsters. Many of the subjects (65.6 per cent) gave no symptoms at all. Of the remainder (34.4 per cent), the following percentages gave the symptoms stated: Cough 53.3; dyspnoea 62.6; expectoration 18.5; haemoptysis 11.6; loss of strength 39.4; loss of appetite 13.9; night-sweats 6.9; fatigue 3.1; and pain in the chest 18.5. The physical signs found on auscultation varied considerably in the different individuals, resembling closely the signs occurring in silicosis. Râles were found in 23.2 per cent of the cases; in 13.4 per cent at the bases of the lungs, in 5.8 per cent at the apices, and in 4 per cent they were scattered. Three cases had semi-fine, persistent râles heard at the apices, after an expiratory cough. In two of these cases the roentgenograms revealed some evidence of active tuberculosis. *Roentgen findings:* A study of the roentgenograms shows a decided enlargement of the hilum shadows in 91.2 per cent of the cases, and a slight enlargement in 7 per cent. The shadows are very dense and in 15 per cent of the cases contain one or more large calcified spots in the hilum. In one patient these spots were so numerous that the entire hilum appeared to be filled. The linear markings, while somewhat more pronounced than in the average metal miner, are not as noticeable as in early silicosis. The markings extend to all parts of the lungs, but are more noticeable in the lower two-thirds, appearing to increase toward the bases. In 16.3 per cent of the patients, "budding," or early mottling along the bronchial tree, was noticed, indicating beginning silicosis. These patients had been working in lead and zinc mines for several years, and the fine mottling was probably due to silica dust. The most characteristic finding was the large number of discrete, dense, shot-like spots scattered over the lungs. An effort was made to count the spots on flat films. The tabulation shows spots as follows: 28.8 per cent of the subjects had less than 25; 27 per cent from 25 to 50; 11.1 per cent from 50 to 75; 7 per cent from 75 to 100; 10.6 per cent from 100 to 200; 9.6 per cent from 200 to 300; 3.6 per cent from 400 to 500; and 1.6 per cent over 500. In 94 per cent of the cases, from 55 to 95 per cent of the spots were located in the bases. They were scattered about equally over the lung area in 6 per cent of the cases. The cases can be divided roughly into two groups,—those with large spots and those with small spots. The large spots vary in size from 4 mm. to 1 cm., averaging about 6 mm. They are not so numerous as the small spots, and appear to be more dense, discrete, and symmetrical. The large spots are mostly in the bases (average 80 per cent). The small spots vary from 1.5 to 5 mm., averaging 3 mm. They are fairly dense, round and uniform. They are occasionally seen against the heart-shadows, where they closely resemble lead shot of about a no. 8 size. A review of the roentgenograms of the clinic shows that 29 of the cases have been returning at various intervals during the past two or three years. One case shows a decrease in the number of spots in the lungs, has gained weight, and is no longer short of breath. The other cases are about the same as when originally examined. The majority of the cases have been reëxamined from six months to one year apart, and in only 2 cases (tuberculosis cases) has any decided change for the worse been noted. Bacteriological examinations of the sputum revealed a fungus in 31 cases. Two types of fungi were identified,—*Aspergillus fumigatus fisheri* and *Aspergillus niger*. Tubercle bacilli were present in only 2 of the 88 cases in which an examination was made of the sputum.—*Miliary Lung Disease Due to Unknown Cause, R. R. Sayers and F. O. Meriwether, Am. J. Roentgenol. and Rad. Ther., March, 1932, xxvii, 337.*—(*W. I. W.*)

Nontuberculous Infiltrations of Lung.—The more or less acute tuberculous infiltrations resemble clinically and roentgenologically pneumonic processes of other aetiology. The differential diagnosis may be very difficult, especially when resorption occurs. No one single symptom or sign, but only the totality of the observation, will lead to a diagnosis. Following upper respiratory infections, acute infiltrations, nontuberculous, of short duration, are frequently seen. If their centre clears first they may produce roentgenological appearances suggestive of excavations.—*Über flüchtige, entzündliche Lungeninfiltrationen nichttuberkulösen Ursprungs, C. Boytinck, Beitr. z. Klin. d. Tuberk., May, 1932, lxxx, 67.*—(*M. P.*)

Circumscribed Pleuritis.—Old localized fibrotic scars of the pleura may cause roentgenological shadows which simulate parenchymal infiltrations. Such lesions have a

totally different clinical significance than in infiltrations, and the proper differential diagnosis is therefore very important. One of the chief points of differentiation is the fact that pleuritic scars do not change their roentgenological appearance over a long period of time.—*Circumscripte pleuritische Restzustände und ihre differentialdiagnostische Bedeutung, F. Kellner, Beitr. z. Klin. d. Tuberk., August, 1932, lxxx, 297.—(M. P.)*

Mediastinal Pleurisy.—Mediastinal effusions may be classified according to the location of the effusion or the type of exudate. The onset is characterized by an acute febrile attack with pain, unproductive cough and dyspnoea. Pressure symptoms result from encroachment upon the trachea, oesophagus, azygos vein, vagus or recurrent laryngeal nerve. Fluoroscopic and roentgenographic examinations are important in the diagnosis. In differentiating tumors and enlarged lymph nodes from mediastinal pleurisy, it should be noted that the former tend to be near the root of the lung while the latter tends to be near the diaphragm. Cysts are more circular in outline and apt to extend to the opposite side. In pericardial effusion the heart can be outlined within a larger shadow. Cold abscesses cast fusiform shadows about the spine, with destruction of the vertebrae. Mediastinal pleurisy may be confused with aneurysm, substernal thyroid, a large pad of fat at the apex of the heart, densely infiltrated extra lobe of lung, or neurofibroma of the vagus nerve. In mediastinal pleurisy the fluid may rupture into a bronchus. Three cases are cited.—*Mediastinal Pleurisy: Case Reports and X-ray Diagnosis, H. Snure, Radiology, May, 1932, xviii, 1028.—(T. O. R.)*

Cholesterol Pleurisy.—A case of cholesterol pleurisy, occurring in a man who had a previous history of pleural effusion following pneumonia, is reported. The patient died of a tuberculous meningitis. The postmortem findings revealed on the surface of the left lung an attached box-like mass, which assumed the shape of the cavity from which it had been removed. This mass was extremely hard; it measured 23 cm. from apex to base, 24 cm. across its convex anterolateral surface at its widest portion and about 14 cm. in its greatest anteroposterior dimension. The wall was hard and gritty. On section, it was found that this mass was a closed cavity, containing 1,400 cc. of a milky fluid in which shimmering particles were seen. The wall was irregular, varying from 6 mm. to 2 cm. in thickness; it was composed of hyalinized fibrous tissue and contained masses of calcareous material and spicules of bone. In the thickened visceral pleura near the apex of the lung, there was a fibrocaseous nodule which measured 1 cm. in diameter.—*Cholesterol—Thorax in Tuberculosis. (Cholesterol Pleurisy): Report of a Case, H. M. Stein, Arch. Int. Med., March, 1932, xlix, 421.—(W. I. W.)*

Pleural Calcification.—The authors report 3 cases in which there was a deposition of calcium after prolonged pleurisy. The calcium content of the blood-serum was increased to 12.8 to 18 mgm. per cent. Stereoscopic pictures showed that the shadows were outside of the lung. It appeared important to the author that the forerunning pleural disease produced degenerative changes in the tissues and tended to cause a diminution of the circulation in these; in addition there was an excess of calcium present in the blood.—*Pleuraverkalkungen, G. Apitz and G. Frischbier, Zeitschr. f. Tuberk., 1931, lx, 310.—(H. J. C.)*

Acute Empyema.—The differences in results, obtained by the common operative procedures used for the treatment of acute empyema, are to be ascribed not so much to the particular type of operation as to the underlying character of the disease that has caused the empyema. It is well known that respiratory infections vary considerably in virulence in different localities and in different years; and with this there is a corresponding variation in the seriousness of the resulting empyemata. Practically all empyemata secondary to respiratory infections are either of the postpneumonic (pneumococcic) type or of the postinfluenzal type. In the former the disease sets in after the pneumonia has subsided, and is much less severe; in the postinfluenzal type, it occurs while the underlying process is still active, and the desperate illness of the patient is obvious. In gross appearance of the exudate and in bacteriological findings the two are likewise distinctive, although in some cases a differentiation may be very difficult. In simple empyema, without other concomitant disease of importance, the prognosis is very good, irrespective of the type of operation performed, but when the disease is complicated by such other conditions as bilateral pneumonia, contralateral empyema, pulmonary abscess, pericarditis, peritonitis, etc., the prognosis varies with the seriousness of the associated conditions. The treatment of empyema before the war was by rib-resection and open drainage; during the war period the mortality of postinfluenzal empyemata, which had

been as high as 60 per cent, was greatly reduced after the introduction of the method of early aspiration, followed, if necessary, by late thoracotomy. The virulence of these infections was distinctly on the wane at this time, and the decrease in mortality is to be ascribed in great part to this, rather than to the introduction of a new method of operation. As a result of the increased interest in empyema during the war, chronic empyema has been practically eliminated in some clinics. In a study of 425 cases in Baltimore and 158 cases in Cincinnati, the mortality, in uncomplicated empyema, was less than 6 per cent, in one group of 60 cases no deaths occurring; in the Baltimore series there was a total mortality rate of 16.5 per cent, and practically all of these deaths were associated with other serious lesions, demonstrable clinically or by necropsy. A comparison of the two series indicates that the thoracotomy with open-drainage method used in one was approximately as effective as the closed aspiration method used in the other. From a year-by-year study of both series it appears that the mortality rate depends primarily on the virulence of the infection for that particular year and locality, and thus with the incidence of associated lesions. In the Cincinnati series (1922–1930), the incidence of postinfluenzal empyema was much greater than in the Baltimore series (1889–1921). Of the three operative methods in common use, namely, repeated aspirations with or without subsequent closed aspiration drainage or thoracotomy, primary closed aspiration drainage, or intercostal incision or rib-resection with open drainage, there may be theoretical reasons for preference, but the practical results in several large series are essentially the same. Since primary rib-resection with open drainage will occasionally cause death from open pneumothorax in the presence of an underlying pneumonia and a mobile mediastinum this method is less preferable than the other two.—*Acute Empyema, G. J. Heuer, J. Thorac. Surg., June, 1932, i, 461.*—(*B. B.*)

Spontaneous Healing of Pyothorax with External Fistula.—A 27-year-old physician, under treatment by artificial pneumothorax for a unilateral tuberculous lesion of one-year duration, developed a hydrothorax which rapidly became purulent. Successive paracenteses were performed, with the evacuation of large quantities of pus in which tubercle bacilli, streptococci, and colon bacilli were identified. Three draining fistulae developed as a result of these manoeuvres. A regimen of heliotherapy, general and local, sulphur baths, and bed-rest was instituted. Within two years the fistulae had closed and the pyothorax and the underlying parenchymal disease were obliterated by a massive fibrosis. Heliotherapy was sporadically continued for two more years, after which time the patient successfully resumed his usual life. No opinion is hazarded as to which of the therapeutic measures employed was the decisive one in securing this rare result.—*Guérison spontanée d'un pyothorax thérapeutique fistulisé a la peau, M. Ollivier, Rev. d. l. Tuberc., April, 1932, xiii, 412.*—(*A. E. M.*)

Pyopneumopericardium.—The author reports on a case of rupture of a tuberculous cavity into the pericardium, developing into a secondary pyopneumopericardium. The observations were of value because the clinical course could be observed from the onset and the postmortem verified the clinical diagnosis.—*Ein Fall von Pyopneumoperikard, G. Langer, Beitr. z. Klin. d. Tuberk., 1931, lxxvi, 597.*—(*H. J. C.*)

Spontaneous Pneumothorax.—A pneumothorax may be produced by entry of air into the pleural cavity as a result of injury or disease leading to perforation of the parietal or visceral pleura or, in rare cases, perforation of the diaphragm. In nontuberculous disease, such as carcinoma, abscess, gangrene or empyema, the prognosis depends largely on the disease. Treatment consists in relieving pressure symptoms by removal of air or aspiration of fluid. Rupture of an emphysematous bleb is probably the cause of spontaneous pneumothorax occurring in apparently healthy persons. No effusion forms, and complete recovery almost invariably occurs. It is not common in general emphysema associated with chronic bronchitis, but occurs from the rupture of a small bleb with a valvular opening to a bronchiole. Rest in bed and the relief of pressure symptoms are the only measures indicated in treatment. Pulmonary tuberculosis is the most common cause of spontaneous pneumothorax. It may be an early sign but usually occurs in advanced lesions. The perforation may quickly heal with the formation of a clear or purulent effusion. The prognosis is grave in empyema and every effort should be made to obtain reëxpansion of the lung. Aspiration of the purulent fluid and irrigation of the pleural cavity are indicated. Phrenicectomy, oleothorax or thoracoplasty are measures frequently used in attempts to obliterate the space and lessen the amount of purulent

fluid. Drainage is only justified as a last resort to relieve symptoms when the pneumothorax cavity cannot be closed by re-expansion of the lung, or, failing this, by thoracoplasty.—*Spontaneous Pneumothorax, L. S. T. Burrell, Tubercle, July, 1932, xiii, 433.*—(*M. E. P.*)

Spontaneous Pneumothorax.—The term *spontaneous pneumothorax* is a misnomer, because disease in the lung, trauma, or a sudden increase of intrapleural pressure is necessary to produce the tear in the lung. The aetiology of a number of cases remains unknown and is not satisfactorily explained by any of the theories that have been advanced. The male sex, the age-period between thirty and forty, and the right side show a slightly higher incidence. The symptoms are not always characteristic and point sometimes more to abdominal than to pulmonary disease. The intrapleural pressures bear no relationship to the degree of functional disturbance or to the gravity of symptoms. It is probable that spontaneous pneumothorax can be caused by a variety of aetiological factors, as, for instance, a circumscribed emphysematous bleb, pulmonary scars, or blood-clots in a bronchus, which interfere with expiration, or, finally, congenital malformation. Four cases are reported in which the aetiology remained unknown. The right side was affected three times. The objective and subjective symptoms were insignificant and of short duration and all patients made an uneventful recovery.—*Der Spontanpneumothorax, H. Huber, Wien. klin. Wchnschr., August 5, 1932, xlv, 991.*—(*K. F.*)

Recurrent Pneumothorax.—A man, 50 years old, had recurrent attacks of spontaneous pneumothorax. Repeated removals of air from the pleural cavity failed to expand the lung; evidently a bronchopleural fistula had been established. After the intrapleural injection of 20 cc. of a 0.5 per cent solution of silver nitrate, a slowly increasing pleural effusion developed which was spontaneously absorbed and was followed by an obliterative pleuritis. This led to a permanent expansion of the lung, and the patient lost all symptoms and has been well and working during the last year following his illness.—*Persistierender Spontanpneumothorax, durch Obliteration der Pleurahöhle ausgeheilt, A. Kenner, Beitr. z. Klin. d. Tuberk., June, 1932, lxxx, 169.*—(*M. P.*)

Bilateral Spontaneous Pneumothorax.—Pneumonoconiosis and spotaneous non-tuberculous pneumothorax present many unsolved problems. A 43-year-old laborer, complaining of slight breathlessness and transient chest pains of three months' duration, presented no significant findings on physical examination except moderate acyanotic dyspnoea. Nevertheless the roentgenogram revealed a bilateral pneumothorax. The intrapleural pressure was 0, −1. On withdrawing a quantity of air the lungs reëxpanded. Roentgenograms now revealed widely disseminated small dense shadows, sharply outlined, somewhat preponderant in the middle portions of each lung, and tending to conglomerate under the clavicles. The apices were clear. Further questioning revealed an attack of "grippe" with "pulmonary congestion" thirteen years previously, and five years of labor with pick and shovel in a quartz gold mine. The patient was under observation for five months. Pneumothorax recurred twice. The first relapse occurred in the course of a cold, when cough and a small amount of sputum appeared for the first time. No tubercle bacilli could be demonstrated in the sputum by smear or guinea-pig inoculation. Small inorganic crystals, probably silicates, were demonstrated, however. The second recurrence followed a mild coughing spell. Throughout, except for moderate breathlessness, the pateint felt remarkably well and remained afebrile. The Wassermann test was negative. On the basis of the history of exposure to silicate dust, the great discrepancy between the clinical signs and symptoms and the X-ray findings, the inability to demonstrate the tubercle bacillus, and the presence of inorganic crystals in the sputum, the diagnosis of pneumonoconiosis rather than miliary tuberculosis was made, although the latter could not be rigorously ruled out. In discussing the differential diagnosis, the absence of pleural adhesions, the slightly elevated intrapleural pressures, the rapid absorption of the gas, the absence of marked effusions (there was a slight amount of fluid for a short time), the two recurrences, the density and precision of the roentgenological shadows are emphasized in support of the decision. Bibliographical research revealed this case of recurrent spontaneous bilateral pneumothorax associated with pneumonoconiosis (silicosis) to be the first of its kind reported.—*Pneumothorax spontané bilatéral à rechutes, silicose pulmonaire et tuberculose miliare, De Leobardy and Pasquet, Rev. d. l. Tuberc., April, 1932, xiii, 375.*—(*A. E. M.*)

Bronchiectasis.—Bronchiectasis is usually associated with some type of previous

inflammation of the lung, such as bronchitis or the bronchopneumonias that result from influenza, measles, whooping cough or typhoid fever, but it is obvious that it can also arise without such "primary" causes, and can be due to developmental defects, such as atelectasis or faulty development of the pleura, resulting in "tying-off" of the bronchi. In any event, it seems that the clinical appearance of the disease is often intimately associated with some type of respiratory infection, even though this may have preceded the clinical manifestations by a considerable time. Atelectasis, both congenital and acquired, probably plays an important rôle in the future development of bronchiectasis. Bronchopneumonias can produce loss of fibroelastic tissue of the bronchi; repeated attacks of bronchitis can result in dilatation from repeated distention due to coughing. The relationship of the tracheobronchial lymph nodes to the development of bronchiectasis is not clear, and there is doubt that retrograde lymph-drainage to the bronchi occurs. Bronchiolectasis may follow severe pulmonary infections in children when the preterminal bronchi are affected. (In the adult the middle and large-sized bronchi are usually affected.) The dilated areas can be of three types: (1) dilated bronchioles, (2) dilated atria and infundibula, and (3) abscess cavities. This condition of bronchiolectasis in children is also known as honeycombed lung, and is thought by some to be the forerunner of the saccular type of bronchiectasis in adults. Thoracic deformities can result in bronchiectasis. It is doubtful if syphilis plays an important rôle, except as a cause of stenosis of the bronchi. The various factors causing bronchiectasis are classified as follows: (1) intrabronchial (including foreign bodies, stones, tumors); (2) bronchial (including membrane changes, changes in the various elements of the bronchial wall, strictures, etc.); (3) peribronchial (including diseased lymph nodes, adhesions, tumors); (4) pulmonary (including the various types of pneumonia, pulmonary fibrosis, tuberculosis, abscess, emphysema, etc.); (5) pleural (including fistula, adhesions, effusions); and (6) deformities (such as rickets). It is important to recognize the distinct entities when, for example, a tumor produces a localized bronchiectasis. A chronic putrid bronchitis may persist for a long time without the formation of dilated bronchi or bronchioles; the distinction can be made by lipiodol injections, but it is essential that complete drainage of the bronchi has been obtained before lipiodol is injected. In some instances, it is advisable to reëxamine the patient on several days to be sure that lipiodol has not been retained in small terminal pockets. In putrid bronchitis, the amount of sputum may equal that seen in bronchiectasis. Bronchiectatic areas can become dry and remain so until a fresh infection lights them up. Schneider divides bronchiectasis into two main types, cylindrical and saccular. Combinations of these occur. D. T. Smith was able to isolate a member of the anaerobic fusospirochetal group in 49 of 60 cases of proved bronchiectasis. These organisms may, of course, be purely secondary invaders (although Smith has produced bronchiectasis in rabbits with members of this group of organism), but the response to neosalvarsan is good, at least with regard to clearing of the foetor even though the amount of sputum is not reduced. Other factors which may be of importance aetiologically are discussed, such as war gas, fungus infections, mediastinal pleurisy, and influenza. Lung abscess and bronchiectasis are frequently associated. With lipiodol injections, it is important to remember that the abscess may not fill with the oil if it is of the narrow-neck type, thus leaving an unfilled area, around which areas of bronchiectatic change are demonstrable. Abscess-formation may be secondary to bronchiectasis; the reverse is likewise true. Bronchiectasis can of course result from tuberculosis. In many cases of pneumonoconiosis, the clinical picture may be that of the coexisting bronchiectasis. All cases of clinical bronchiectasis are associated with lung abscess, even though the latter may be only microscopic. For convenience a division is made into cases with and without abscess, the former including the cases in which abscess-formation is conspicuous. Of 182 cases, 33 showed definite evidence of abscess-formation. At the time of observation, most of the patients were under 40 years, and in many the onset of the disease occurred 15 or 20 years previously. The two sexes are affected equally. Eighty-three of 149 cases were unilateral, including cases of congenital bronchiectasis. Bronchiectasis is essentially a disease of the lower lobes. The chief symptoms of bronchiectasis are cough, which may be persistent, expectoration, the chief characteristic of which is its foetor, haemoptysis, which is a frequent occurrence and at times fatal, and fever, associated often with chills and sweats, usually indicating some type of a "pneumonitis." The presence of fever does not necessarily indicate mere retention in the bronchi, but often represents the effects of parenchymal involvement. Pain is not common. Gastrointestinal symptoms suggest the existence of amyloid disease. Skin eruptions

are rare. Joint pains are frequent. In the diagnosis of bronchiectasis, one should be able to locate the lesion, determine the size and distribution of the dilatations, as well as the degree of atelectasis and of mediastinal shift, the aetiological factors, and the presence of such features as abscess, bronchial stenosis, tumor, amyloid disease, etc. Bronchography, bronchoscopy, and diagnostic pneumothorax are valuable diagnostic procedures, although the latter is but little used since the introduction of lipiodol. In the physical examination, changes in râles with change in posture are of significance. The blood-count may show a moderate anaemia, and a varying leucocytosis. The presence of albumin in the urine should raise the suspicion of amyloid disease which makes any operative procedure dangerous. Layering of the sputum is inconstant; the presence of Dittrich plugs is significant; eosinophiles are not numerous in the sputum. No diagnosis of bronchiectasis is complete without lipiodol injection, which in most adults can be done by the Singer aspiration method. Examination of the patient under the fluoroscope, before and after lipiodol injection, is of value in determining the location of the lesion, the presence of adhesions, and the type of breathing. In children suffering from bronchiolitis and bronchiolectasis, expiratory dyspnoea, with a low-lying diaphragm, is often seen. Lipiodol injection, while of great value, is not a perfect diagnostic procedure in so far as it does not always demonstrate the extent of the lesions, especially of some abscesses, nor does it designate the localization of the more active portions of the lesion. Bronchoscopy can be of great aid in localizing the suppurating areas, ruling out foreign bodies, permitting more complete lipiodol injections, and in obtaining pure cultures of the lesions. The well-known association of nasal-sinus infections with bronchiectasis is discussed, and 8 case reports are given, in only one of which did distinct clinical improvement result from treatment of the sinuses. In bronchorrhoea from nasal-sinus infections, without bronchiectasis, marked improvement can result from successful sinus treatment. At the present time, there is no real proof that nasal-sinus infection can produce bronchiectasis, nor *vice versa*. Certainly, however, the association between the two diseases is common. A cure of bronchiectasis can only occur when the diseased tissue is removed or destroyed. Clinical remissions are frequent, but are usually not of long standing. Spontaneous cures occur very rarely. The anatomical lesion, once present, never heals. Of the various forms of treatment, rest, diet, climate, heliotherapy, and the thirst cure are not thought to be of any definite value. Neoarsphenamine, when used in cases showing fusiform bacilli and spirochaetes, at times gives striking results; in other cases it is completely ineffectual. No lasting improvement was obtained in any case by its use alone. Postural drainage is by far the most satisfactory of the simpler methods of treatment, used continuously or intermittently. Bronchial irrigations, use of superheated air with drug mixtures, and vaccine therapy are of no real value. Repeated lipiodol injections, as a therapeutic procedure, have, in some patients, produced temporary alleviation of symptoms, but in no case has actual improvement of the lesion resulted. The beneficial effects of such lipiodol injections are thought to be due to mechanical replacement of the retained material rather than to an iodine effect, although it has been shown that the iodine content of the blood rises quickly after injection of lipiodol into the lung. Bronchoscopy has a definite place in the treatment of bronchiectasis, especially in cases due to partial bronchial stenosis. Repeated aspirations, or even a single aspiration, often relieves symptoms strikingly. As far as pneumothorax is concerned few patients with true bronchiectasis respond to pneumothorax treatment even though collapse may be almost complete; it rarely does harm by promoting retention of secretions, as one might expect. When supplemented by postural drainage and neosalvarsan, pneumothorax can be of value in early cases, but not apparently in chronic cases. Oleothorax seemed to benefit one case; in the other in which it was tried, it was unsuccessful. Bilateral pneumothorax has not been used extensively; bilateral phrenicectomy or combinations of phrenicectomy and pneumothorax have not been used frequently enough to decide about their value. Opinions about treatment by plombage must be held *sub judice*, but the results so far reported seem promising. Thoracoplasty has been performed by a number of surgeons but the total number of cases is small. According to Hedbloom, it is not to be done in cases complicated by multiple abscesses, as indicated by septic temperature, leucocytosis, etc. Of the 32 patients subjected to thoracoplasty by Hedbloom, 21 are still alive and in many of these appreciable improvement has occurred. Following a poor result with thoracoplasty, lobectomy can then be performed. Phrenicectomy for bronchiectasis is being done with increasing frequency, although the authors in summarizing the literature and 26 cases of their own conclude that an individual with bronchiec-

tasis has only a relatively small chance of being improved by the operation, and, on the contrary, a definite chance of being made worse. Moreover, even when improvement occurs it is usually not lasting. Of the total of 82 cases analyzed, 22 showed definite improvement late, 9 were moderately improved, 9 were slightly improved, and 24 were not improved. Eight were made worse, and the result was uncertain in seven. Pneumotomy in the treatment of bronchiectasis has been discarded, since with modern diagnostic methods we know that the lesions are usually multiple and drainage of one area is more or less useless. Cautery pneumonectomy, the method of Graham, has been performed with good results. It is indicated in those cases of bronchiectasis associated with multiple abscesses which have not responded to simpler forms of therapy and in which thoracoplasty or lobectomy cannot produce satisfactory results. In extensive bilateral disease and in the saccular type of bronchiectasis (perhaps of congenital origin) cautery pneumonectomy seems contraindicated. The technique of the operation is described in detail. It consists essentially of rib-resection, removal of the underlying periosteum, and then (usually in a later stage or in multiple stages) superficial cauterization by a soldering-iron of the involved lung area for the purpose of producing external drainage from all of the areas of bronchial involvement. Bronchial fistulae naturally result, but it is surprising how many of these heal completely, and, even though they remain open, how little discomfort they produce. Of 54 cases of chronic lung suppuration the late results (over 4 years of observation) showed 36 patients alive and working, 34 patients who had made definite improvement, and 6 operative deaths. Bronchial fistulae were still present in 17 cases. These reports include cases of lung abscess and bronchiectasis. Bettman had successful results with this method in 4 out of 5 cases (one case of bronchiectasis, four of abscess). There are 212 recorded cases of lobectomy performed for bronchiectasis, of which death from operation has occurred in 72 patients. In 99 cases a thoroughly satisfactory result was obtained. These figures include all the reported cases, but if one considers only the more recent reports, the mortality from operation is about 15 per cent, and, of the remaining cases, excellent results are obtained in 65 per cent. In view of the serious aspects of the disease, however, it is apparent that this procedure is of distinct value. Many of the individual reports are discussed in this article, which also presents the operative technique employed by various surgeons, consisting usually of a two-stage operation, with ligation of the lobe (or part of a lobe), followed by excision, or gangrene development with subsequent sloughing. There has been one case of bilateral cautery pneumonectomy reported; Eloesser is reported to have done a successful bilateral lobectomy for bronchiectasis. In bronchiectasis, the prognosis of an individual untreated case is uncertain. It is true that the disease is not always progressive, but there is no evidence that true bronchiectasis can spontaneously disappear. The fact that the saccular dilatations of the congenital variety frequently are manifested first in adult life indicates that bronchial dilatations may exist for years without producing symptoms. If the bronchiectasis results from a foreign body, the chances of a favorable outcome vary indirectly with the delay with which the body is removed, although a foreign body in a lung, even though it remains a long time, does not always produce bronchiectasis. When atelectasis and fever are present, the prognosis is usually worse than when parenchymal involvement does not exist. The existence of numerous peribronchial abscesses, seen at times in children following one of the exanthemata, often carries with it a poor prognosis. The presence of intranuclear inclusion bodies in pertussis, recently demonstrated, with destruction of the bronchial wall may be correlated with the subsequent development of bronchiectasis in these children. The most unfavorable type of bronchiectasis in children is that associated with mediastinal shift to the affected side; many of these cases have erroneously been diagnosed as having unresolved pneumonia. Except for those cases due to tumors or tuberculosis, mediastinal shift in the adult is uncommon. Haemoptysis in adults can occur in cases of "dry" bronchiectasis, not associated with cough or expectoration; these cases are easily confused with tuberculosis. Chronic empyema is one of the common complications of bronchiectasis. Amyloid disease is a serious complication since it renders the necessary radical operative procedures much more dangerous. Brain abscess is also a not uncommon complication, but the explanation for the association of the two is not clear, since it has not been proved that the cerebral lesion occurs by direct metastasis *via* the heart. The correction of such factors as foreign bodies and strictures, more care during convalescence from the bronchopneumonias of pertussis and the exanthemata, and earlier attempts at clearing up nasal-sinus infections may be of aid in preventing the development of bronchiectasis, but at present there is no prophylactic measure

that carries with it any assurance that its employment will avoid the development of this disease. An extensive bibliography is appendid to this series of four articles.—*Bronchiectasis, H. Ballon, J. J. Singer and E. A. Graham, J. Thorac. Surg., December, 1931, i, 154; February, 1932, i, 296; April, 1932, i, 397; June, 1932, i, 502.*—(*B. B.*)

Bronchial Stenosis.—Although bronchial stenosis is in many ways comparable to any constriction found in other hollow viscera, it has not been well studied clinically; the introduction of bronchoscopy and lipiodol into the study of bronchial diseases has added many facts to our knowledge of bronchial stenosis, but these facts have not as yet been well organized. Stenosis of the bronchi can be classified as being congenital, due to compression of the tracheobronchial region or of the larger bronchi, or to intrinsic factors in these areas due to inflammatory changes, or those that are essentially restricted to the bronchioles. Of the congenital anomalies, some of which are expressed by bronchiectasis, one of the common defects is an unusually high or unusually persistent Cuvier's duct, which may produce obstruction to the whole lung or, if acting as an obstruction only later in development, of the left lower lobe. The rapidly growing chest-wall and diaphragm, acting on a maldeveloped, nonexpansile lung may result in a uni- or multiocular cyst, termed *bronchiectasis universalis*. Or the area of involvement may be localized, and the cysts that result can press on a bronchus. Such a case was described by Rach, in which a large cyst, lined by ciliated epithelium, but not communicating with a bronchus, compressed the right main bronchus. The types of congenital abnormalities of the lung are numerous, depending on the stage of lung development in which they occurred, some being traceable as far back as the pulmonary bud. At times portions of lung tissue become separated, forming mediastinal cysts. In a man of twenty who had a drainage for what was thought to be empyema, lipiodol demonstrated a constriction of the left main bronchus below which a bilocular formation was seen, at the summit of the diaphragm, communicating with two bronchi. At operation, cysts of the lower lobe were found and drained, but a large cyst found in the upper lobe was untouched; the patient made a good recovery. In another patient, congenital cystic disease of the left lung produced marked retraction of the mediastinum and contraction of the lung. An incidental finding in another patient was a large cyst of the right lower lobe which did not fill on lipiodol injection. If the cyst is air-containing, there must be incomplete obstruction; complete obstruction produces atelectasis or mucous cysts. A large number of causes of compression stenosis are evident, including goitre, tumors, cysts, dilatations of adjacent viscera such as the heart and the aorta, mediastinitis, lymphadenitis, and scars. Pressure from a large thymus may produce compression of the trachea and sudden death, but does so rarely, and then probably only in infants. In mitral stenosis, the left main bronchus can be compressed. Inflammatory process of the mediastinal lymph nodes can compress, distort or even perforate bronchi. Chronic mediastinitis, without lymphadenitic swelling, can produce difficulty in breathing, as well as in swallowing. In two cases presented, calcified hilum nodes apparently produced bronchial stenosis, and one of these patients died from an acute haemorrhage. In another patient with inspiratory stridor and deviation of the trachea anteriorly, mediastinotomy revealed the cause to be a large chain of tuberculous peritracheal nodes. Tuberculous lymphadenitic abscesses occur at the top of the sternum as well as in the tracheobronchial region. Other types of mediastinal infiltrates, as well as exudates, mediastinal pleurisies, and empyemas can produce stenosis. Schmorl has described *bronchitis deformans*, a sclerotic peribronchitis and lymphadenitis in the aged, which can result in stenosis. Intrinsic stenoses are produced by foreign bodies, tumors, intramural inflammatory masses and their scars, and by softenings of unknown origin. Tracheal carcinoma is rare. Chondroösteoplastic tracheopathy, a disease of unknown origin, associated with the formation of bony or cartilaginous ridges, is not uncommon. Syphilitic scars are more common in the upper portions of the tracheobronchial tract, although the lower bronchi may be involved by this disease. Submucous tuberculous infiltrations may be extensive enough to cause stenosis; sclerosis from tuberculous peribronchitis usually involves bronchi of the second order. A tracheomalacia of unknown origin has been described. Most factors affecting the larger bronchi, such as tumors, aneurysm, etc., produce stenosis of one side or of one lobe. Stenosis of the middle bronchi, from the second or third order down, is usually due to a foreign body or a diffuse sclerosing process. Pneumonoconiosis and tuberculosis, or the two together, often involve bronchi of the third or fourth order. The "asthma" of the tuberculous may be more often due to the emphysema resulting from such processes than to asthmatic hypersensitiveness. Swelling

and destruction of the mucosa of the bronchioles can be caused by inhalations of gases, and also occur after influenzal infections and the infectious bronchitides. Such changes have been described under the term *bronchiolitis fibrinosa*, and represent one of the end-results of war-gas inhalation. From physiological studies it is apparent that a satisfactory understanding of the changes that occur with tracheal stenosis has not been reached. Partial stenosis of the trachea produces inspiratory dyspnoea, in which inspiration is prolonged, and is deeper and more forceful. With this there occurs retraction of the interspaces, decrease of pleural pressure, and increase of the manometric readings of the intrapleural oscillations between inspiration and expiration. Since expiration is passive, no expiratory dyspnoea occurs in relatively mild stenosis. The lung thus becomes dilated by active inspiration. The characteristic stenotic type of breathing disappears after vagotomy, and in diabetic coma, and begins, in the presence of obstruction, long before changes in the blood gases can be demonstrated; the stenotic type of breathing, then, is induced by nervous changes, in which the higher cortical centres may play an important rôle. If obstruction becomes sufficient to cause expiratory dyspnoea more striking mechanical changes occur, since the ordinary mechanism of expiration is almost entirely passive. Acute dilatation of the lung occurs, with resultant decompensation of the respiratory and circulatory systems, and peripheral venous stasis. This condition, which is often associated with collapse, can be overcome either by the pressure of the air behind the obstruction becoming great enough to overcome the latter, or by drop in the blood-pressure sufficient to release the constriction. The effects of severe dyspnoea from stenosis are thus quickly transmitted to the peripheral circulation. The pulmonary arterial pressure does not rise in stenosis, as has previously been thought. Dilatation of the heart occurs in some stages of inspiratory dyspnoea, and diminution in size in expiratory dyspnoea. In a patient with emphysema, asthma, and a tracheal obstruction in whom expiration was prolonged, exercise produced a reversion of the type of breathing, with lengthening of inspiration and decrease of expiration time. The variability of bronchial stenosis in causing dilatations of the bronchi is difficult to understand, nor is it clear why some bronchial obstructions will produce changes in the peripheral portions of the bronchi without causing emphysema. Inflammatory infiltrations of the bronchi and the pliability of the lung tissue under the influence of the pull exerted by the chest-wall are two factors influencing this selectivity. If the stenosis becomes complete atelectasis will result. In intermittent stenosis marked shiftings of the mediastinum can occur. Tuberculosis, syphilis, rhinoscleroma, leprosy, typhoid fever, actinomycosis, and other infections can produce stenosis of the trachea and bronchi. In tuberculosis this occurs in 4 per cent of all cases, excluding those with laryngeal involvement. Depending on the location of the stenosis, the type, whether acquired or congenital, the presence or absence of infection, and the completeness of the stenosis, varied types of pulmonary pathology can occur, including cyst-formation, abscess-formation, pleuritis, etc. The symptoms of stenosis have been divided by Gerhardt into the irritative stage, the period of constant dyspnoea, and the period of suffocative attacks. In high stenosis and in stenosis at the bifurcation the stridor is loud; when only one of the main bronchi is stenosed, dyspnoea is less severe and is not characterized by the stages mentioned. In one-sided lesions, lying on the good side may set off an attack. If one lobe only is involved there may be no dyspnoea. In diffuse stenosis dyspnoea may be severe. Cough is a fairly constant symptom; it is brassy and unproductive when the obstruction is high. The watery sputum that sometimes is expectorated may be due to distal atelectasis and oedema. A thin copious sputum alternating with a dry cough is suggestive of intermittent stenosis. Difficulty in swallowing may result from immobility of the oesophagus rather than from intrinsic lesions of the oesophagus. Dyspnoea, stridor and inspiratory retraction of the intercostal spaces are evident in stenosis of the trachea or of the bifurcation. Normal resonance of the chest, or hyperresonance, is present; breath-sounds are diminished, as a rule, but may be tubular or amphoric; rhonchi and râles may be found. Congestion of the peripheral veins, cyanosis, and enlargement of the right heart may be present, and liver enlargement may be found. Pupillary differences may be noted, pointing to sympathetic irritation; hoarseness from laryngeal palsy may exist. Tracheobronchoscopy should always be performed in these cases of high obstruction. Plain X-rays may be valuable in determining the underlying process, but usually lipiodol is required to demonstrate the exact abnormality of bronchial configuration and position. When a major bronchus is occluded signs of massive collapse develop, including shrinking of the chest, immobility of that side, shifting of the mediastinum toward the side involved and marked eleva-

tion of the diaphragm, which often shows paradoxical movement. If the occlusion is recent, Jacobson-Holzknecht's phenomenon may be evident, that is, inspiratory shift of the mediastinum toward the affected side, and expiratory shift back to the midline. The obstructed chest becomes dull or flat, and the breath-sounds are much diminished or absent. Dyspnoea is ordinarily not excessive. On X-ray, the affected lung is opaque, that side of the chest is shrunken, the mediastinum shifted, and occasionally the cause of the obstruction is visible, such as tumor, etc. A heavy exposure for the affected side is recommended to bring out underlying lesions not visible in plates taken with ordinary exposures. The injection of lipiodol, 5 to 15 cc., should be done under the fluoroscope with small quantities at a time so that the oil can be watched in its passage, rather than depending on plates taken at the end of the injection. The exposure for the lipiodol chest plates should be heavier than for the routine chest picture. In intermittent unilateral stenosis, the changes that occur are often striking, and at times confusing. A man, 28 years of age, had had two attacks of pneumonia and then an attack of chills, fever, and unproductive cough. A stenosis of the left main bronchus was demonstrated, and, following a preliminary pneumothorax, an exploratory thoracotomy was done, when enlarged hilum nodes were found on the right side. These were not removed, but later examinations showed that the stenosis had disappeared. Six months later he died after an attack, with chills and high fever, and autopsy showed a distinct stenosis. Two cases of syphilitic stenosis are presented. Various types of stenosis, total, partial or intermittent, undoubtedly occur much more commonly than generally suspected. Lipiodol injection is not advisable in tuberculosis because of the danger of spread of infection, but bronchoscopy should be done in cases of questionable stenosis in tuberculosis. In one such case, phrenic-nerve evulsion was done immediately after the diagnosis of tuberculous stenosis was made by bronchoscopy. Stenosis of a single lobe or less may produce retraction of the corresponding chest-wall, especially if it involves a lower lobe. Bronchoscopy is less satisfactory in the diagnosis of upper-lobe than in lower-lobe lesions. Lipiodol is of great help in diagnosis of stenosis if the proper technique is used, although it is difficult at times to obtain satisfactory pictures of the upper lobes. In diffuse stenosis of the smaller bronchi, diagnosis may be difficult; the symptoms are more characteristic than the signs. The patients have constant dyspnoea with alarming acute exacerbations, the chest is flat, the respiratory rate is increased, the intercostal spaces retract in all areas on inspiration, the breath-sounds may be diminished, diffuse coarse râles are heard, the cough is often distressing, the sputum is watery and frothy, the extremities are often cyanotic, the pulse is rapid and small, the blood-pressure low, the right heart may be enlarged. It is important to differentiate oedema of the glottis and laryngeal obstruction from lower stenosis, especially because of the indications for tracheotomy. In laryngeal stenosis the head is thrown back; in tracheal stenosis the chin is thrust forward, the head downward. Respiratory excursion of the larynx is less in tracheal than in laryngeal stenosis. The aetiological differential diagnosis of stenosis is discussed. A case is cited in which the differential diagnosis between a malignant lung tumor and tuberculous lymph nodes pressing on a bronchus was most difficult. Treatment of tracheal stenosis and stenosis at the bifurcation should begin with bronchoscopy unless the patient is in an acute attack or a dangerous aneurysm exists. Old scars may be dilated with a bougie. Syphilitic stenosis should be treated with mercury, but not with iodides because of the possibly fatal increase of secretion. The debris of an ulcerating tuberculous lymph node may be removed with forceps or by aspiration; mild X-ray treatment is of value in treating enlarged tuberculous lymph nodes, although great care is required if a parenchymal lung lesion exists. When the stenosis arises from a posterior mediastinal abscess, drainage should be established by posterior drainage. High stenosis from an aneurysm is usually hopeless, but relief has been obtained by decompressive mediastinotomy, in which portions of the upper sternum and the first, second, and third costal cartilages are removed. In stenosis due to removable growths, treatment is obvious. In other types of tumors, such as Hodgkin's disease or lymphosarcoma, stenotic symptoms occur late, and are usually due to venous congestion rather than to actual tumor pressure on the bronchial tree. Mediastinotomy is of little help in these cases; fortunately, many of them are sensitive to X-ray. In the treatment of tumors or abscesses causing high obstruction, anterior mediastinotomy has been successfully done. For unilateral stenosis the indications for emergency relief do not usually exist. Tuberculous stenosis is most common in the region below the bifurcation, either from enlarged hilum lymph nodes or from old scars. Syphilis and tumors are rare here as causes

of stenosis, except for large inoperable tumors. Operative access to this region is considerably more difficult than to the region of the trachea or its bifurcation. One must penetrate the pleura to reach masses at the hilum; the operation is difficult, and only successful when well-encapsulated masses are present and removable. Artificial pneumothorax, by allowing the mediastinum to return to its normal position and by straightening kinks in the bronchi, may be of considerable value especially in relieving distressing symptoms, although it is not aimed directly at overcoming the stenosis. It is conceivable in some cases of tuberculosis in which thorocoplasty produces an unexpected febrile reaction that the underlying cause of the exacerbation is a bronchial stenosis, and for this reason it may be wise, in those cases in which a stenosis is suspected, to bronchoscope the patient before operating. In stenosis of a bronchus supplying a single lobe, the most common cause is a foreign body; next in frequency is carcinoma. When bronchoscopy alone is not successful in allowing the foreign body to be removed, artificial pneumothorax followed by bronchoscopy should be tried; if these fail pneumotomy must be resorted to. When a tumor is located in the proximal portion of a bronchus, just before it enters the lung parenchyma, it can be removed through an incision into the bronchus, after the lobe has been delivered and turned medially; if the tumor is not movable, the bronchial wall is undoubtedly involved, and lobectomy should be performed. If the site of the bronchial tumor is intraparenchymal, as is usually the case, lobectomy is indicated. The technique for lobectomy is described, and the importance of great care in preventing bleeding, and watching for it, is stressed. Transfusions are often of great value postoperatively, but one should be careful to differentiate haemorrhage from dilatation of the right heart and impairment of the lesser circulation, under which circumstances transfusion is contraindicated. Some of the other causes of lower stenosis, such as inflammatory masses, aneurysm and mitral stenosis, preclude lobectomy; except in cases of aneurysm, dilatation should be attempted. For drainage of a stenotic lobe, cauterization is done, preceded, if necessary, by an adhesion-forming operation; with the subsequent retraction of tissues, more suppurating areas may come into vision, requiring repeated cauterizations. The resulting bronchial fistulae may remain open indefinitely; if expectoration ceases entirely and the patient's condition warrants, it may then be closed by extirpating the remains of the shrunken lobe.—*Bronchial Stenosis, L. Eloesser, J. Thorac. Surg., December, 1931, i, 194; February, 1932, i, 270; April, 1932, i, 373; June, 1932, i, 485.*—(*B. B.*)

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